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Acute Emergency: A Case Report of a Carotid Artery Dissection

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Abstract

Background

Carotid hematoma is a rare occurrence in critically ill hospitalized patients. However, there is a four percent chance of development of a carotid hematoma leading to a carotid artery dissection [11]. Associated with ear, nose, and throat (ENT) surgeries.

Methods

Case study reviewing a patient who developed a carotid hematoma, which led to carotid artery dissection and required emergency surgery. The patient's hospital course including lifesaving interventions is discussed.

Summary

In order to reduce mortality in patients who develop this complication, early recognition, airway management, medical treatment, and surgery are critical to survival.

Keywords

Carotid hematoma, carotid artery dissection, surgical emergency, ENT.

Carotid Artery Dissection: A case of a ruptured carotid hematoma Overview

Carotid hematoma is a rare but significant occurrence in critically ill hospitalized patients [9]. The incidence peaks in the fifth decade of life and is equally represented in both sexes [7]. However, there is a four percent chance of ear, nose, and throat (ENT) surgeries in which a carotid hematoma may lead to a carotid dissection from trauma, tumor, and friability of the artery [11]. Carotid artery dissection occurs in approximately 2.6 cases per 100,000 [7]. In order to reduce mortality among this patient population, early recognition, airway management, medical treatment, and surgery are critical to survival [9].

This case study highlights this rare complication: a carotid dissection, which occurred following a laparoscopic neck dissection surgery. A carotid hematoma resulted from a tear during surgical dissection of a neck tumor. After dissection of the carotid artery, aggressive and emergency treatment and surgical intervention ensued. The hospital course requiring lifesaving interventions is discussed, including pathophysiology of carotid hematoma formation and dissection, clinical features, diagnosis, and treatment.

Pathophysiology

The development of a carotid hematoma is strongly correlated with a hemorrhage within the wall of the vessel, typically localized to the medial layer [12]. There are primarily two sources for the hemorrhage: an intimal tear dissecting into the media or a primary hemorrhage of the vasa vasorum into the media [12]. Outcomes of the hematoma are based on the location and the extent of hemorrhage.

Hematomas can lead to narrowing of the intimal wall, occlusion of the vessel, dilatations of the vessel, or pseudoaneurysm formation [12]. This alteration to the vessel wall becomes a thrombogenic surface on which platelets aggregate causing a cascade of events leading to thrombus or hematoma formation [7].The timing from initial intimal tear to formation of a hematoma leading to dissection is highly variable in different individuals, which may explain the delayed onset and variety of presentations and symptoms [12].

Factors which may predispose an individual to a carotid hematoma leading to dissection include: tumors, radiation near the vessel, previous surgery, positioning during intubation or extubation, chronic infection, accidental trauma, Marfan syndrome, coronary artery disease, uncontrolled hypertension, predisposing blood dyscrasias, intimal defects, or they may occur spontaneously[3]. The unusual stress or injury to the vessel causes a collection of blood and platelet attraction and aggregation. This is a precipitating factor leading to a hematoma formation. The additional damage caused by the hematoma can lead to a pseudoaneurysm, an outpouching from the original vessel, stenosis, or additional strain ultimately causing an intimal tear or complete dissection [12].

Radiation may cause fibrosis of the vessel and surrounding tissues precipitating friability leading to activation of the coagulation cascade resulting in hematoma formation [5]. Tumors and previous surgery with manipulation near or including the vessel can cause occlusion of the vessel invoking inflammation and aggregation of platelets at the site of injury [7].

Once a hematoma has formed in the vessel, the vessel is compromised and susceptible to further injury.The events inducing carotid dissection are suggestive of the velocity of blood flow through the vessel, thrombogenesis, and the extent of the damage that the vessel undergoes [8].

Clinical Features

Common clinical symptoms which can occur with a carotid artery dissection consist of: pain at the site upon initial tear, headache or head pain, development of changes in level of consciousness, a transient loss of vision also known as amaurosis fugax, transient ischemic attack (TIA), stroke, or suffocation [12]. Additional symptoms, which may be present, also include: neck swelling, dyspnea, and orthopnea [6]. Ipsilateral Horner's syndrome may also be present upon assessment of carotid artery dissection [15]. Horner's syndrome includes miosis ipsilaterally to the dissection, sluggish pupillary reflexes, sinking of the eye into the cavity also known as enophthalmos, a vascular headache, and moderate eyelid ptosis caused by carotid dissection [15].

Diagnosis

The diagnosis of carotid artery dissection is based on the clinical presentation, patient history, and angiography.

The patient's past and current medical history are valuable factors, which can help narrow the differential. A patient with a history of uncontrolled hypertension would have had recurrent damage to the vessel leading to increased velocity of blood flow contributing to vascular wall abnormalities [8]. After surgical removal of a tumor that is in close proximity to the vessel or radiation therapy to the site of tumor growth, the vessel is predisposed to fragility and friability.

There are several diagnostic tests that can be performed to identify the presence of a carotid artery dissection. These include: ultrasonography, computerized tomography (CT) and CT angiography (CTA) [12]. The gold standard, however, is magnetic resonance imaging (MRI), or magnetic resonance angiography (MRA) [12].

Ultrasound has only been shown to be approximately 75-85% predictive of dissections and percentages of specificity are unknown [1]. The use of ultrasonography fails to detect carotid dissection by location alone. The internal carotid artery lies behind bones and this increases the difficulty of detection via ultrasonography [13].

The use of CT and CTA are used more specifically helpful in certain populations where an MRI or MRA is contraindicated, such as in those with pacemakers, retained foreign objects (metal, screws or plates), or shunts implanted during elective surgeries [14]. Magnetic resonance angiography at this time remains the best method for exclusion of carotid artery dissection [14].

Magnetic resonance imaging and MRA are more commonly being used to diagnose carotid artery dissection. In carotid artery dissection MRA displays hyperdense signals on T1 weighted images [15]. The use of MRI may be restricted in use to those patients who are not claustrophobic [12]. Allergies to contrast may also preclude both CTA and MRA [13]. The sensitivity of these exams is highest two days after the initial dissection [15]. Both MRI and MRA assist in the determination of the age of the hematoma by the signal intensities seen on both T1 and T2 images [4]. This may also determine the further course of action. If the dissection appears to be long standing then medical management may be considered, whereas in an acute dissection more aggressive therapy would need to be indicated [4].

Treatment

Acute carotid artery dissection constitutes a medical emergency and requires airway management and immediate surgical repair [5]. As in most emergencies, airway management is the first priority [9].

In the case of carotid dissection, it is especially critical due to the location of the lesion as it is life threatening and because failure to secure the airway prevents further definitive treatment [12]. Blood pressure management and blood product administration may need to be given to maintain a homeostatic euvolemic state depending upon the severity of the dissection.

Blood pressure management is critical to the viability of organs being perfused as well as to prevent continued bleeding and further dissection of the vessel [9]. Blood pressure management is individualized based on the presentation of the patient and the symptoms [4]. The treatment and management of blood pressure may be variable from using vasopressive medications for hypertension to the use of blood pressure lowering medications for hypotension [9]. A balance between adequate vital organ perfusion and limiting the dissection must be reached so that latent effects of dissection are not seen later after repair [9].

The mainstay of treatment in a dissecting carotid artery includes: control of embolization and prevention of further thrombus formation [7]. Depending upon the size and severity of the hematoma and dissection, either medical management or surgical intervention may be appropriate.

Medical management is comprised of antiplatelet therapy with or without anticoagulation therapy [7]. Antiplatelet and anticoagulation therapy is controversial in the use of carotid dissection [7]. They may be part of the medical management started one to two days after surgical repair of the dissection to prevent further thrombus formation [7]. Patients without an acute dissection are typically monitored for disease progression with frequent diagnostic tests such as an MRI. However, if further dissection occurs or increased neurological deficits are apparent then emergency repair of the vessel is needed [7]. This may increase the degree of complexity of surgical dissection around the dissecting vessel.

Surgical repair may generate inflammation at the site of repair. Repair may induce further platelet aggregation, contributing to postsurgical complications [7]. Therefore, symptomatic presentation and diagnostic laboratory values are critical to initial and continued management of the patient. Further disease progression with hemodynamic instability or deteriorating neurological symptoms would necessitate emergency surgical intervention [7].

Management following surgery

Prognosis is determined by control of hemostasis and recanalization of the dissected artery [7]. Carotid artery dissection results in approximately 40% mortality [10]. Following successful surgical intervention patients need to be admitted to an intensive care unit. Maintenance of airway management and hemodynamic stabilization are key components to the postoperative care.

Case Study

T.C. is a 54-year-old male who required a neck dissection for removal of a malignant tumor. Despite his surgeon's advice that an open neck dissection would allow better access and limited risk, T.C. requested the use of a laparoscopic approach because it would provide a more aesthetically pleasing result. After explaining the risks and benefits of the laparoscopic surgery, the surgeon obtained informed consent. Upon arrival to the surgical intensive care unit (SICU) after surgery, T.C. was intubated with a 7.5 French endotracheal tube (ETT) and sedated with a midazolam drip. No vasopressive management was needed at the time of admission. He was hemodynamically stable and all vital signs were within normal limits. His wife and her best friend were at the bedside.

Less than four hours postoperatively, a small slow trickle of blood appeared out of T.C.'s right nostril. The ear, nose, and throat (ENT) resident was paged to inform him of the change in status.

Upon examination of T.C., profuse amounts of blood were being expelled via T.C.'s bilateral nares and out of his ETT without resolve. T.C. was found to be in distress with continued profuse bleeding. During this time T.C. became hemodynamically unstable and the initiation of vasopressive treatment began. Eight units of uncrossed matched packed red blood cells (PRBC) and platelets were given intravenously prior to surgical intervention. A Propofol intravenous infusion was started to control the violent coughing and gagging. Suctioning of bilateral nares and ETT inline suctioning were continuously performed. T.C. was subsequently transported to the operating room (O.R.).

T.C. has a history of malignant neoplasm of his tongue with involvement of his neck. He had previously undergone radiation to shrink the tumor prior to surgical removal. He has a 30-pack history of tobacco use including cigarettes and cigars. He has no other significant past history or family history.

Hospital Course

Upon examination under anesthesia in the O.R. a hematoma and right carotid artery dissection were found to be the sources of bleeding. An emergency radical neck dissection, tracheostomy placement, and evacuation of hematoma were successfully completed. A total of six additional units of PRBCs and three units of platelets were infused during surgery. A total of 1500 mls of blood loss was estimated during the surgical procedure. An arterial line placed in his right radial artery, a triple lumen central line in his left subclavian vein, and central venous pressure monitoring were placed in the O.R.

Immediately after emergency surgery T.C. was transported back to his SICU room. He was hemodynamically stable at this time. He remained on a Propofol intravenous infusion for sedation, fentanyl drip for pain control and phenylephrine for blood pressure control. He was reconnected to the ventilator and placed on assist control (A/C) with a rate of 12, tidal volume (Vt) 550, pressure support (PS) of 10 and peep of 5. He was to remain sedated and on the ventilator until the next morning.

Serial labs were drawn on T.C. every four hours to include a complete blood count (CBC), basic metabolic panel (BMP), magnesium, phosphorus, ionized calcium, arterial blood gases, and lactic acids. Lab values were not able to be accessed during review and case presentation and have been omitted for this purpose. Assessment of the surgical site and prior area of hematoma formation were to be completed hourly for the first twenty-four hours after surgery.

On postoperative day two, phenylephrine was weaned off. The mean arterial pressure (MAP) goal of 65-70mm Hg was maintained over the course of twenty-four hours. T.C. remained stable throughout the remainder of the day. His central venous pressure fluctuated between eight and ten mmHg. During this time anticoagulation therapy was started with heparin 5000 units subcutaneously every twelve hours and a sequential compression device (SCD) was applied and maintained for 22 out of 24 hours a day.

On postoperative day three Propofol was weaned off. Ventilator settings remained stable adequately ventilating and oxygenating T.C. The site of hematoma formation and carotid artery dissection were without edema, redness, soft to touch, and without indications of hematoma formation. His CVP fluctuated between six to eight mmHg.

On postoperative day six the patient was transferred to the step-down unit (SDU) where he stayed until discharge without the need of additional emergency management until day fifteen. T.C. was discharged home fully recovered and decannulated.

Initial Postoperative Problems

Carotid Artery Dissection

Dissection of a carotid artery is not a common occurrence [9]. Approximately only 2.6 cases out of 100,000 result in carotid dissection (Jaipersad et al., 2012).

The most common cause of mortality is the lack of ability to correctly identify the diagnosis and inadequate airway management [15]. Less than four hours postoperatively from T.C.'s initial laparoscopic neck dissection, he began to bleed profusely. The severity of T.C.'s blood loss prior to emergency surgery was concerning. Although he had an ETT in place, a secure airway was not maintained until anesthesia and a paralytic agent were given prior to the commencement of surgery. He required multiple vasopressors prior to emergency surgery and rapid blood product administration. Based on the knowledge of the attending ENT physician who had prior experience as a resident, he hypothesized the patient was having symptoms of a ruptured carotid hematoma with possible carotid artery dissection.

Discussion

T.C. was a significant case of carotid artery dissection and hematoma formation. The initial emergency surgery was successful. His postoperative course was without additional need of emergency management. The tear in the intimal vessel led to hematoma formation and dissection. Fundamental to T.C.'s success was expeditious assessment, airway management, and emergency surgical management. While angiography may be the gold standard to detect dissection, time was of the essence, and this could not safely be performed. By performing an open radical neck dissection, the carotid artery was identified, and diagnosis of a hematoma and carotid artery dissection were made quickly.

Summary

Carotid artery dissection is a rare but significant clinical occurrence in critically ill hospitalized patients occurring both spontaneously and traumatically [9]. Assessment of preoperative risk factors, assessment of potential complications throughout the hospitalization, and communication are instrumental in the continuum of care for patients undergoing tumor removal by neck dissection whether the repair is performed laparoscopically or with open surgical intervention. Early diagnosis, airway management, diagnostic interventions, and emergency surgery are essential to decrease mortality associated with carotid artery dissection and improve patient outcomes [9]. Successful lifesaving resuscitation measures are dependent upon early recognition and facilitation of prompt treatment

References

1. Benninger, D. H., & Baumgartner, R. W. (2006); Ultrasound diagnosis of cervical artery dissection. Frontal Neurology Neurosciences, 21, 70-84.
2. Carper, E., & Haas, M. (2006); Advanced practice nursing in radiation oncology. Seminars in Oncology Nursing, 22(4), 203-211. doi:10.1016/j.sonen.2006.07.003
3. Carvalho, K. S., Edwards-Brown, M., & Golomb, M. R. (2007); Carotid dissection and stroke after tonsillectomy and adenoidectomy. Pediatric Neurology, 37(2), 127-129.
4. Hab, M., Pfefferkorn, T., Cyran, C. C., Grimm, J., Rominger, A.,et al, (2011); Age determination of vessel wall hematoma in spontaneous cervical artery dissection: A multi-sequence 3T cardiovascular magnetic resonance study. Journal of Cardiovascular Magnetic Resonance, 13(76), 1-8.
5. Hanakita, S., Iijima, A., Ishikawa, O., Kamada, K., & Saito, N. (2011); Treatment of a cervical carotid pseudoaneurysm that occurred years after laryngectomy and irradiation of a neck tumor. The Japan Neurological Society, 51, 588-591.
6. Harjai, K. J., & Anderson, J. (2008); Repair of right carotid artery pseudoaneurysm in a patient with hostile aortic arch through left ventricular approach. Journal of Interventional Cardiology, 21(3), 239-241.
7. Jaipersad, A. S., Tiivas, C., Walton, G., & Imray, C. H. (2012); A novel treatment for embolizing carotid dissection. International Journal of Surgery Case Reports, 3(1), 19-21.
8. Okamoto, T., Miyachi, S., & Yoshida, J. (2005); Animal models of cervical artery dissection. Frontiers of Neurology and Neuroscience, 20, 1-11.

9. Pazardzhikliev, D. D., Yovchev, I. P., & Zhelev, D. D. (2008); Neck Hematoma Caused by Spontaneous Common Carotid Artery Rupture. The Laryngoscope, 118(4), 684-687.
10. Powitzky, R., Vasan, N., Krempl, G., & Medina, J. (2010); Carotid blowout in patients with head and neck cancer. The Annals of Otology, Rhinology, & Laryngology, 119(7), 476-484.
11. Rao, A. S., Makaroun, M. S., Marone, L. K., Cho, J. S., Rhee, R., & Chaer, R. A. (2011); Long-term outcomes of internal carotid artery dissection. Journal of Vascular Surgery, 54(2), 370-374.
12. Stahmer, S. A., Raps, E. C., & Mines, D. I. (1997); Carotid and Vertebral Artery Dissections. Emergency Medicine Clinics of North America, 15(3), 677-698.
13. Suzuki, R., Koga, M., Toyoda, K., Uemura, M., Nagasawa, H., Yakushiji, Y.,...Minematsu, K. (2011). Identification of internal carotid artery dissection by transoral carotid ultrasonography. Cerebrovascular Diseases, 33(4), 369-378.
14. Taschner, C. A., Leclerc, X., Lucas, C., & Pruvo, J. P. (2005); Computed tomography angiography for the evaluation of carotid artery dissections. Frontal Neurology Neurosciences, 20, 119-128.
15. Thanvi, B., Munshi, S. K., Dawson, S. L., & Robinson, T. G. (2005); Carotid and vertebral artery dissection syndromes. Postgraduate Medical Journal, 81(956), 383-388.